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JENELL LAWSON
INTELLECTUAL PROPERTY
CURAGEN CORPORATION
555 LONG WHARF DRIVE
NEW HAVEN, CT 06551

EXAMINER	
SULLIVAN, DANIEL M	

ART UNIT	PAPER NUMBER
1636	

DATE MAILED: 06/01/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/804,014	LI ET AL.	
	Examiner	Art Unit	
	Daniel M. Sullivan	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *21 January 2005 and 17 March 2005*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *5,9,10,12-14,30,33 and 44-46* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *5, 9, 10, 12-14, 30, 33 and 44-46* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

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DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 21 January 2005 has been entered.

This Office Action is a response to the 21 January Paper filed in response to the Final Office action mailed 21 September 2004. Claims 5, 9, 10, 12-14, 30, 33 and 44-46 were considered in the 21 September Office Action. Claim 10 was amended in the 21 January Paper. Claims 5, 9, 10, 12-14, 30, 33 and 44-46 are pending and under consideration.

Response to Amendment

Claim Rejections - 35 USC § 102

Claim 10 stands rejected under 35 U.S.C. 102(b) as being anticipated by NCBI online, Accession No. AC008687 for reasons of record. Applicant has amended the claim such that it is now directed to a nucleic acid molecule comprising a nucleotide sequence encoding a polypeptide comprising an amino acid sequence having one or more conservative substitutions to SEQ ID NO: 8. As the claim requires only that the nucleic acid encode "an amino acid sequence" having one or more conservative amino acid substitutions, the claim can reasonably construed as encompassing a nucleic acid encoding any amino acid sequence (*i.e.*, any two more amino acids) of the sequence set forth as SEQ ID NO: 8. As described in the previous Office Actions, the

sequence disclosed in AC008687 comprises regions having substantial identity with the instant SEQ ID NO: 7, which encodes SEQ ID NO: 8, and is 100% identical from nucleotides 904 to 1692 (see especially Appendix A of the Paper filed 3 July 2003). Thus, absent evidence to the contrary, the sequence disclosed in Accession No. AC008687 encodes a sequence of at least two amino acids having one or more conservative substitutions to SEQ ID NO: 8 as required by the instant claim 10.

Claim Rejections - 35 USC § 112

Claims 5, 9, 10, 12-14, 30, 33 and 44-46 stand rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement for reasons of record and herein below in the response to Applicant arguments.

New Grounds

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claim 10 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Vas-Cath Inc. v. Mahurkar, 19USPQ2d 1111, clearly states that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession *of the invention*. The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed.*" (See page 1117.) The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See *Vas-Cath* at page 1116).

In the instant case, the claim is directed to an isolated nucleic acid molecule comprising a nucleotide sequence encoding a polypeptide comprising an amino acid sequence having one or more conservative substitutions to SEQ ID NO: 8. With regard to conservative substitutions, the specification, at page 54, teaches:

> In addition to naturally-occurring allelic variants of the NOVX sequence that may exist in the population, the skilled artisan will further appreciate that changes can be introduced by mutation into the nucleotide sequence of SEQ ID NO: 1, 3, 5, 7, 9, 11, 13, 15, 17, 19 or 21, thereby leading to changes in the amino acid sequence of the encoded NOVX protein, without altering the functional ability of the NOVX protein. For example, nucleotide substitutions leading to amino acid substitutions at "non-essential" amino acid residues can be made in the sequence of SEQ ID NO: 1, 3, 5, 7, 9, 11, 13, 15, 17, 19 or 21. A "non-essential" amino acid residue is a residue that can be altered from the wild-type sequence of NOVX without altering the biological activity, whereas an "essential" amino acid residue is required for biological activity. For example, amino acid residues that are conserved among the NOVX proteins of the present invention, are predicted to be particularly unamenable to alteration.

Thus, the nucleic acid of the claim, even if it were limited to comprising a nucleotide sequence encoding a polypeptide comprising an amino acid sequence having one or more conservative substitutions to the sequence set forth as SEQ ID NO: 8, the nucleic acid can encode any modification of SEQ ID NO: 8 does not "alter the functional ability of the NOVX protein", wherein the nature of a "functional ability" is not defined by the specification.

The Guidelines for Written Description state: "when there is substantial variation within the genus, one must describe a sufficient variety of species to reflect the variation within the genus" (Federal Register, Vol. 66, No. 4, Column 3, page 1106). "The written description requirement for a claimed genus may be satisfied through sufficient description of a representative number of species by actual reduction to practice..., reduction to drawings..., or by disclosure of relevant, identifying characteristics, i.e., structure or other physical and/or chemical properties, by functional characteristics coupled with a known or disclosed correlation between function and structure, or by a combination of such identifying characteristics, sufficient to show the applicant was in possession of the claimed genus" (MPEP §2163(3)(a)(ii)).

In the instant case, there is clearly substantial variation within the claimed genus, which encompasses any nucleic acid that encodes any protein having the "functional ability" of the NOV4 protein. The specification discloses a detailed description of a single nucleic acid within the claimed genus, *i.e.*, a nucleic acid consisting of SEQ ID NO: 7, and teaches that it encodes a polypeptide that is structurally similar to a family of voltage gated potassium channel proteins. However, the teachings of the specification fail to establish a correlation between the structural characteristics of a NOV4 protein and the "functional abilities" of the NOV4 protein such that the skilled artisan would have recognized that Applicant was in possession of the broad genus of any nucleic acid that encodes any protein having the "functional ability" of a NOV4 protein.

In view of these considerations, a skilled artisan would not have viewed the teachings of the specification as sufficient to show that the applicant was in possession of the claimed invention commensurate to its scope because it does not provide adequate written description for the broad class of nucleic acids claimed. Therefore, only the described nucleic acid encoding the

polypeptide set forth as SEQ ID NO: 8 meets the written description provision of 35 U.S.C. §112, first paragraph.

Claim Rejections - 35 USC § 101

Claims 5, 9, 10, 12-14, 30, 33 and 44-46 are rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific or substantial asserted utility or a well-established utility. Although the claims were not previously rejected under 35 USC §101, upon further consideration of the record as a whole it is apparent that the disclosure fails to assert a specific and substantial utility for the claimed invention and the disclosed properties of the claimed nucleic acid do not do not support a well-established utility.

The pending claims have been reviewed in light of the Utility Examination Guidelines and Guidelines for Examination of Patent Applications under 35 U.S.C. §112, first paragraph, "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1092-1111, Friday, January 5, 2001

The Examiner is using the following definitions in evaluating the claims for utility.

"Specific"-A utility that is *specific* to the subject matter claimed. This contrasts with a *general* utility that would be applicable to the broad class of the invention.

"Substantial"-A utility that defines a "real world" use. Utilities that require or constitute carrying out further research to identify or reasonably confirm a "real world" context of use are not substantial utilities.

"Credible"- Credibility is assessed for the perspective of one of ordinary skill in the art in view of the disclosure and any other evidence of record that is probative of the applicant's assertions. That is, the assertion is an inherently unbelievable undertaking or involves implausible scientific principles.

"Well-established"-a specific, substantial and credible utility which is well known, immediately apparent, or implied by the specification's disclosure of the properties of a material alone or taken with the knowledge of one skilled in the art.

The claimed subject matter is not supported by either a specific or substantial asserted utility because the disclosed utilities are generally applicable to a broad class of molecules and the specification fails to set forth the unique properties of the claimed invention such that the skilled artisan would recognize a specific real-world utility therefor.

The claims are directed to a nucleic acid molecule encoding the human polypeptide set forth as SEQ ID NO: 8, which has significant structural homology with a family of voltage gated potassium channels. The specification asserts that the nucleic acid is useful as a marker for human chromosome 19 and for therapeutic application in Episodic Ataxia, type 1, Long QT Syndrome 1 and 2, Benign Neonatal Epilepsy, Jervell and Lange-Neilson syndrome, Autosomal dominant deafness (DFNA 2), non-insulin dependent diabetes mellitus, CNS disorders, arrhythmia, seizure, asthma, hypertension therapy and/or other pathologies and disorders. The specification further asserts that the nucleic acid can be used for identification of therapeutics which modulate the channel and therefore modulate insulin secretion.

However, the teachings with regard to the asserted utilities are not specific to the claimed invention or are based on unsubstantiated properties of the nucleic acid. The specification (beginning at page 16 and continued through page 21) teaches that the polypeptide having the sequence of SEQ ID NO: 8 shares significant homology with a family of voltage-gated potassium channels and thus would credibly have the function of a voltage-gated potassium channel and the specification teaches that nucleic acids encoding the polypeptide set forth as SEQ ID NO: 8 can be used to produce the protein, to raise antibodies, to detect mRNA, to detect genetic lesions, to modulate activity of the protein and to screen for drugs or compounds. However, the specification fails to teach to what purpose the nucleic acid, or protein and

antibodies produced therefrom, will be applied. The specification suggests that the claimed nucleic acid can be used as marker for chromosome 19; however, this is not a patentable utility because it is not specific to the claimed nucleic acid. With regard to treatment or diagnosis of one of the multitude of diseases contemplated in the specification, the disclosure provides no teaching that would indicate that the NOV4 protein or nucleic acid could be used as a therapeutic reagent, or is a target for therapeutic target in any of those diseases. The only asserted utility for the reagents developed using the claimed invention is therapeutic, yet the specification provides no specific guidance as to how the agents developed with the invention can be used therapeutically. All of the teachings in the specification related to therapy are general in nature and based on circumstantial evidence. Therefore, the skilled artisan seeking to use the claimed invention therapeutically or in the identification or production of therapeutics would have to experiment to confirm that the nucleic acid could actually be used as contemplated.

Applicant has previously argued that all members of the Kv family share the common utility of transporting potassium ions. However, this is neither a specific nor substantial utility because there are a wide variety of proteins capable of transporting potassium ions, each of which will have a distinct utility based on the unique properties of the protein. For example, one would not assert that the sodium-potassium ATPase, the target of cardiac glycosides used in treating heart disease, has the same specific and substantial utility as the stomach hydrogen-potassium ATPase, which is the target for agents used to treat gastroesophageal reflux disease. Thus, an assertion that a protein is capable of transporting potassium ions is not a specific and substantial asserted utility.

Applicant has also cited Kalman *et al.* (1998) *J. Biol. Chem* 273:5851-5857 as teaching a mouse polypeptide having some structural similarity to the protein encoded by the instant nucleic acid which is expressed in heart, skeletal muscle and pancreatic islet cells, and a protein mapped to a disease locus. Applicant asserts that it is well known in the art at the time that potassium channels are involved in neuromuscular disorders. Applicant also cites art indicating that another protein related to the *Drosophila* Shaker family of K^+ channels is expressed in pancreatic islets and insulinomas. Applicant concludes from this that, "it is clear to a person skilled in the art that the NOV4 polypeptide is involved in the transport of potassium ions and that the NOV4 polypeptide is associated with pathologies including diabetes mellitus and neuromuscular disorders such as acquired neuromyotonia."

These arguments appear to assert that teachings from the art establish a role for the polypeptide encoded by the claimed nucleic acid in diabetes mellitus and acquired neuromyotonia such that the skilled artisan could use the claimed invention to identify modulators which could then be used to treat diabetes mellitus and neuromuscular disorders without undue experimentation. However, as discussed at length in the previous Office Actions, the art recognizes that polypeptides having the structure of a voltage gated potassium channel could play a role in any one of a variety of physiological or pathological conditions and that, given the state of the art, one of ordinary skill would not know what the physiological or pathological function of a polypeptide would be based only on its structural similarity to the family of voltage gated potassium channels. The art cited by Applicant in prosecution fails to support a well-established utility for the claimed nucleic acid. Although Kalman *et al.* speculates that the potassium channel described therein "may contribute at least one subunit to

hetromulitimeric Kv channels in pancreatic β cells" (paragraph bridging the left and right columns on page 5856) the skilled artisan would not know how to treat diabetes mellitus using a modulator of the protein Kalman *et al.* is actually referring to, let alone a modulator of any protein having limited structural similarity to that protein. In fact, before one could use the claimed invention to develop modulators as potential therapeutics to treat diabetes mellitus it would have to establish that the polypeptide plays a role in the disease, which would require empirical experimentation to reasonably confirm.

Likewise, Vincent *et al.* (2000) *Eur. J. Biochem.* 267 :6717-6728, cited by applicant as teaching that potassium channels are involved in neuromuscular disorders, teaches that the voltage gated potassium channel or channels responsible for neuromyotonia has not yet been established (see especially the first full paragraph on page 6724). Therefore, again the skilled artisan would have to establish that the polypeptide encoded by the claimed invention actually played a role neuromyotonia, or any other neuromuscular disorder, before one could use the invention to screen for potential therapeutics for those conditions.

As discussed in previous Office Actions, the asserted utilities for the claimed nucleic acid appear to be based on its encoding a polypeptide that is likely a voltage gated potassium channel and physiological functions established for other members of the family. However, the art teaches that there are many different proteins belonging to the family of voltage gated potassium channels. These proteins are expressed in different tissues and are involved in various physiological and path physiological processes.

Christie *et al.* (1995) *Clin. Exp. Pharmacol. Physiol.* 22:944-951 (previously made of record) teaches that Kv channels in mammals are structurally and functionally diverse and are

differentially distributed among different cell groups (see especially the section entitled "Structural diversity of Kv channels in mammals" beginning on page 946, and the paragraph bridging pages 948-949). Wickenden (2002) *Pharmacol. Ther.* 94:157-182 (previously made of record), in discussing the state of the art with regard to potassium channels as therapeutic drug targets, teaches that the physiological role of potassium channels in pathologies is extraordinarily complex and unpredictable. In particular Wickenden teaches that different tissues contain different complements of potassium channels which may or may not contribute to a disease sate or therapeutic intervention. For example, Wickenden teaches, *inter alia*, that Kv1.3 channels are found in lymphocytes and contribute to lymphocyte activation (section 6.1.1), that there are a several different types of potassium channels found in cardiac cells some of which may contribute to long QT syndrome (e.g., h-ERG (section 6.2.1) or KCNQ1 and KCNE1 (section 6.2.2)) and some of which may be targets for treatment of atrial fibrillation (e.g., Kv1.5 (section 6.2.5)), that altered distribution of Kv1.1 and Kv1.2 may be responsible for axonal dysfunction in conditions associated with demyelination (see section 6.3), that CNS neurons are endowed with many kinetically and pharmacologically distinct potassium currents (see section 6.4), that Kv4.2 may enhance synaptic plasticity (see section 6.4.4), that Kv1.1 may be involved in partial seizures in humans (see section 6.4.4) and that the channel primarily responsible for insulin secretion from pancreatic β-cells is I_{KATP} (see section 6.4.4). Finally, Wickenden teaches, "a combination of biophysical, pharmacological, and genetic approaches has started to provide an understanding of the molecular composition of many important native K^+ channels ...Much work remains to be done, however, before the full potential of K^+ channel modulators can be realized. It will be particularly important in the post-genome era to understand what role each K^+

channel gene product plays in the formation of native currents and what role each molecularly defined K^+ current plays in cellular physiology and pathophysiology" (paragraph bridging the left and right columns on page 171).

Viewed as a whole, the teachings from the relevant art indicate that polypeptides having the structure of a voltage gated potassium channel could play a role in any one of a variety of physiological or pathological conditions and that, given the state of the art even as of 2002 (when Wickenden was published), one of ordinary skill would not know what the physiological or pathological function of a polypeptide would be based only on its structural similarity to the family of voltage gated potassium channels. Therefore, based on the teachings of the art, the skilled artisan would not know what conditions within the unlimited list contemplated in the specification might be amenable to treatment or diagnosis according to the teachings of the specification. Given the art recognized functional diversity, the skilled artisan would be unable to use the claimed nucleic acid, or other reagents developed therefrom, for the utilities asserted in the specification without first engaging in empirical experimentation to reasonably confirm that the claimed NOV4 protein actually could be used as contemplated.

Thus, in view of the record as a whole, it is clear that the teachings of the specification fail to provide a utility for the claimed nucleic acid that is both specific and substantial and fail to disclose the properties of the invention such that a specific and substantial utility would be immediately apparent to one of ordinary skill in the art.

Applicant should explicitly identify a specific and substantial credible utility for the claimed invention and establish a probative relation between any evidence of record and the originally disclosed properties of the claimed invention.

Response to Arguments and the Declaration under 37 C.F.R. §1.132

Applicant's arguments and the showings of the declaration are now addressed as they apply to the rejection under 35 USC §112, first paragraph, set forth in previous Office Actions and the 35 USC §101 rejection set forth herein.

In the paragraph bridging pages 4-5 of the 21 January Paper, Applicant contends that the specification enables one of ordinary skill in the art to use the claimed nucleic acids to identify therapeutics that modulate the Kv channel function of the polypeptide of SEQ ID NO: 8, thereby treating disorders such as diabetes or neuromuscular disorders. The arguments, which were addressed in previous Office Actions, are reiterated in the Declaration by Dr. Daniel Rieger.

The Declaration, at paragraph 5, cites Kalman *et al.* as teaching that a mouse protein having 83% identity with the instant NOV4 is expressed in heart and skeletal muscle with high expression levels. In paragraph 6, the Declaration points out that methods of identifying molecules that can modulate functions of an identified ion channel are taught in the specification and were well known in the art at the time of filing. Based on this, Declarant concludes, "a person skilled in the art can readily perform a cell-based assay...to identify molecules that can modulate the functions of the potassium channel of the invention. Such modulators are <u>likely</u> useful as therapies for the treatment of disorders <u>such as</u> diabetes or neuromuscular disorders" (bridging page 2-3 of the Declaration; emphasis added).

These arguments have been fully considered but are not deemed persuasive. First, it should be made clear that an opinion as to a legal conclusion made in a Declaration is not entitled to any weight (MPEP §716.01(b)). Thus, the arguments are probative only to the extent

that the basis for the opinion is considered in view of the record as a whole. As stated in the previous Office Action in response to arguments based on the teachings of Kalman *et al.*, Applicant appears to be asserting that teachings from the art establish a role for the polypeptide encoded by the claimed nucleic acid in diabetes mellitus and acquired neuromyotonia such that the skilled artisan could use the claimed invention to identify modulators which could then be used to treat diabetes mellitus and neuromuscular disorders without undue experimentation. As discussed at length in the previous Office Action and herein above, the art recognizes that polypeptides having the structure of a voltage gated potassium channel could play a role in any one of a variety of physiological or pathological conditions and that, given the state of the art even as of 2002 (when Wickenden (*supra*) was published), one of ordinary skill would not know what the physiological or pathological function of a polypeptide would be based only on its structural similarity to the family of voltage gated potassium channels. The art cited by Applicant is no more enabling for the claimed nucleic acid than is the specification itself. Although Kalman *et al.* speculates that the potassium channel described therein "may contribute at least one subunit to hetromulitimeric Kv channels in pancreatic β cells" (paragraph bridging the left and right columns on page 5856) the skilled artisan would not know how to treat diabetes mellitus using a modulator of the protein Kalman *et al.* is actually referring to, let alone a modulator of any protein having limited structural similarity to that protein. In fact, before one could use the claimed invention to develop modulators as potential therapeutics to treat diabetes mellitus it would have to establish that the polypeptide plays a role in the disease, which would require undue empirical experimentation. Therefore, the teachings cited by Declarant provide fail to support an enabled patentable utility for the claimed invention.

In paragraphs 7-9, Declarant cites data submitted with the Declaration as Exhibit 1 indicating that the nucleic acid encoding the polypeptide is highly expressed in skeletal muscle and contends that the claimed nucleic acid can be used to differentiate skeletal muscle form other tissue types such as to distinguish a lung metastasized cancer of skeletal muscle origin form normal lung tissues.

The showings of the Exhibit and Declarant's arguments have been fully considered but are not deemed persuasive in view of the record as a whole. First, Applicant is reminded, evidence to supplement a specification which on its face appears deficient under 35 U.S.C. 112 must establish that the information which must be read into the specification to make it complete would have been known to those of ordinary skill in the art. (*In re Howarth*, 654 F.2d 103, 210 USPQ 689 (CCPA 1981)). Affidavits or declarations presented to show that the disclosure of an application is sufficient to one skilled in the art are not acceptable to establish facts which the specification itself should recite. (*In re Buchner*, 929 F.2d 660, 18 USPQ2d 1331 (Fed. Cir. 1991)). See MPEP 716.09.

In the instant case, the specification provides no information regarding expression of the claimed nucleic acid in skeletal muscle and the art does not disclose any information directed to the expression or utility of the instantly claimed invention. Clearly, therefore, the information which must be read into the specification in order to make it complete would not have been known to those of ordinary skill in the art and the facts established by the declaration should have been, but were not recited in the specification. It is worth noting that the specification at page 21 lists several specific conditions to be treated using therapeutics developed using the claimed invention, such as diabetes, asthma, Long QT syndrome and a variety of CNS disorders,

yet the data submitted in Exhibit 1 indicate little or no expression in pancreas, lung, heart, or any CNS tissue or organ. Thus, the data fail to support the utilities actually contemplated in the specification. Still further, Applicant's assertion that the claimed nucleic acid can be used to differentiate skeletal muscle form other tissue types such as to distinguish a lung metastasized cancer of skeletal muscle origin form normal lung tissues is neither specific nor substantial. Normal skeletal muscle cells are readily distinguished from other cell types based on both histological characteristics and expression of genes such as alpha actin. Furthermore, the Declaration does not provide any data obtained from a cancer of skeletal muscle origin and, because the expression of many genes is altered in cancer cells relative to their cell type of origin, expression of the claimed nucleic acid in cancer cells would have to be established experimentally to confirm that the invention could be used to distinguish a cancer of skeletal muscle origin from any other cell type.

Applicant's arguments and the showings of the Declaration have been fully considered but are not deemed persuasive in view of the record as a whole. Therefore, the claims stand rejected under 35 USC §101 and 112, first paragraph, as lacking a patentable enabled utility.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Daniel M Sullivan whose telephone number is 571-272-0779. The examiner can normally be reached on Monday through Friday 6:30-3:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Remy Yucel, Ph.D. can be reached on 571-272-0781. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Daniel M. Sullivan, Ph.D.
Examiner
Art Unit 1636